

Beyond the Single Molecular Target

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Abstract

This concept paper examines beyond the single molecular target. It develops an argument drawn from Beyond Targets, identifies the relevant biological or clinical mechanisms, and considers where that argument may fail. This is a narrative perspective, not a systematic review or an original clinical study.

The central problem

Single-target pharmacology has delivered antibiotics, hormone replacement, receptor-specific therapies and major oncology advances. The novel proposal is not that precise targets fail; it is that certain chronic phenotypes arise from networks with redundancy, compensation and interactions that can limit the effect of changing one node. A therapeutic target and a disease mechanism are not always the same level of explanation.

Mechanisms and causal architecture

Drug action propagates through physiological networks. Blocking one receptor can change feedback hormones, central appetite signaling, autonomic output and downstream gene expression. Some modern drugs are already multisystem interventions despite being called targeted—for example, GLP-1 receptor agonism engages pancreatic, gastrointestinal and central pathways. Conversely, broad-spectrum drugs can produce greater unwanted effects. The important distinction is whether the intervention's observed benefits align with causal network changes, not whether it is rhetorically “holistic.”

A discriminating example

GLP-1 pharmacology is instructive: one receptor system participates in effects on appetite, gastric emptying and pancreatic hormone signaling. This does not mean the drug corrects every underlying metabolic abnormality, nor that multi-target botanical blends outperform it. Instead, it demonstrates how molecular specificity and systems consequences can coexist. An alternative systems theory should explain successes of modern therapeutics as carefully as their limitations.

Competing explanations and limitations

A failure to cure does not imply wrong therapeutic scale. Many chronic diseases involve permanent tissue damage, ongoing exposure or inherently incurable processes. Reducing myocardial infarction risk or preventing seizures without eradicating a predisposition is substantial clinical success. Moreover, introducing multiple pharmacological agents multiplies interaction risks, and a sophisticated systems narrative cannot substitute for evidence of efficacy.

What evidence would change the interpretation?

A meaningful systems claim should nominate distinct interacting modules, specify anticipated response to perturbation, and predict which combinations or sequences will outperform standard treatment. Simple molecular models remain preferred when they explain the data with fewer assumptions. The goal is greater causal resolution, not ideological opposition to targeted medicine.

Implications for medical interpretation

The article is an invitation to evaluate therapeutic architecture alongside molecule choice. Target precision and systems awareness need not be rivals; in practice they may be complementary.

Selected scientific reading

Barabási, Gulbahce & Loscalzo (2011), Network medicine: a network-based approach to human disease, Nature Reviews Genetics.

<https://doi.org/10.1038/nrg2918>

Drucker (2018), Mechanisms of action and therapeutic application of GLP-1, Cell Metabolism.

<https://doi.org/10.1016/j.cmet.2018.03.001>

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